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Original Research

Assessment Of Serum Potassium Levels In Acute Myocardial Infarction Patients: An Observational Study

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ABSTRACT

Background: One of the leading causes of death across the world is Acute myocardial infarction (AMI). Alteration in serum potassium levels is known to occur in patients with AMI. Hence; we planned the present study to assess the serum potassium levels in AMI patients.

Materials & methods: The present research was conducted on 30 AMI patients and 30 healthy controls and it involved assessment and comparison of serum potassium levels in all the subjects. Blood samples were taken and sent to the laboratory for the evaluation of serum potassium levels. Auto-analyser was used for assessment of serum potassium levels. All the results were recorded and analysed by SPSS software. **Results:** Mean serum potassium levels of the subjects of the AMI group was 4.5 mEq/L while mean serum potassium levels of the subjects of the control group was 4.1 mEq/L respectively. Non-significant results were obtained while comparing the mean serum potassium levels among subjects of the control group and the AMI group. **Conclusion:** Electrolyte disturbance might contribute towards the pathophysiology of AMI.

Key words: Acute myocardial infarction, Potassium

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INTRODUCTION

One of the leading causes of death across the world is Acute myocardial infarction (AMI). Acute myocardial infarction can be divided into two categories, non-ST-segment elevation MI (NSTEMI) and ST-segment elevation MI (STEMI).¹⁻³ Unstable angina is similar to NSTEMI. However, cardiac markers are not elevated. Among patients suffering from acute myocardial infarction, 70% of fatal events are due to occlusion from atherosclerotic plaques. Atherosclerotic rupture leads to an inflammatory cascade of monocytes and macrophages, thrombus formation, and platelet aggregation. This leads to the decreased oxygen delivery through the coronary artery resulting in decreased oxygenation of the myocardium.⁴⁻⁶ Inability to produce ATP in the mitochondria leads to the ischemic cascade, and therefore apoptosis (cell death) of the endocardium, or myocardial infarction. Alteration in serum potassium levels is known to occur in patients with AMI.^{7,8} Hence; we planned the present study to assess the serum potassium levels in AMI patients.

MATERIALS & METHODS

The present research was conducted on 30 AMI patients and 30 healthy controls and it involved assessment and comparison of

serum potassium levels in all the subjects. Written consent was obtained from all the patients after explaining in detail the entire research protocol. Complete demographic details of all the subjects were obtained. Exclusion criteria for the present study included:

- Patients with history any other systemic illness,
- Patients with any known drug allergy,
- Patients with any other underlying metabolic disorder

All the subjects were called in the morning time and blood samples were obtained. Blood samples were sent to the laboratory for the evaluation of serum potassium levels. Auto-analyser was used for assessment of serum potassium levels. All the results were recorded and analysed by SPSS software. Chi-square test was used for assessment of level of significance. P- value of less than 0.05 was taken as significant.

RESULTS

A total of 60 subjects were included in the present study. Among these 60 subjects, 30 were controls while the remaining 30 were AMI patients. Mean age of the patients of the AMI group and the control group was 36.1 years and 38.7 years respectively. There were 20 males in the AMI group while there were 18 males in the control group. Mean serum potassium levels of the subjects of the

AMI group was 4.5 mEq/L while mean serum potassium levels of the subjects of the control group was 4.1 mEq/L respectively. Non-significant results were obtained while comparing the mean serum potassium levels among subjects of the control group and the AMI group.

Table 1: Details of the patients

Parameter	AMI group	Control group
Number of subjects	30	30
Mean age (years)	36.1	38.7
Males	20	18
Females	10	12

Table 2: Comparison of serum potassium levels among AMI group and control group

Parameter	AMI group	Control group	P- value
Serum potassium levels (mEq/L)	4.5	4.1	0.52

DISCUSSION

In the present study, mean serum potassium levels of the subjects of the AMI group was 4.5 mEq/L while mean serum potassium levels of the subjects of the control group was 4.1 mEq/L respectively. Non-significant results were obtained while comparing the mean serum potassium levels among subjects of the control group and the AMI group. Goyal A et al determined the relationship between serum potassium levels and in-hospital mortality in AMI patients in the era of β -blocker and reperfusion therapy. All patients had in-hospital serum potassium measurements and were categorized by mean postadmission serum potassium level (<3.0, 3.0-<3.5, 3.5-<4.0, 4.0-<4.5, 4.5-<5.0, 5.0-<5.5, and \geq 5.5 mEq/L). Hierarchical logistic regression was used to determine the association between potassium levels and outcomes after adjusting for patient- and hospital-level factors. There was a U-shaped relationship between mean postadmission serum potassium level and in-hospital mortality that persisted after multivariable adjustment. Compared with the reference group of 3.5 to less than 4.0 mEq/L (mortality rate, 4.8%; 95% CI, 4.4%-5.2%), mortality was comparable for mean postadmission potassium of 4.0 to less than 4.5 mEq/L (5.0%; 95% CI, 4.7%-5.3%), multivariable-adjusted odds ratio (OR), 1.19 (95% CI, 1.04-1.36). Mortality was twice as great for potassium of 4.5 to less than 5.0 mEq/L (10.0%; 95% CI, 9.1%-10.9%; multivariable-adjusted OR, 1.99; 95% CI, 1.68-2.36), and even greater for higher potassium strata. Similarly, mortality rates were higher for potassium levels of less than 3.5 mEq/L. In contrast, rates of ventricular fibrillation or cardiac arrest were higher only among patients with potassium levels of less than 3.0 mEq/L and at levels of 5.0 mEq/L or greater. Among inpatients with AMI, the lowest mortality was observed in those with postadmission serum potassium levels between 3.5 and <4.5 mEq/L compared with those who had higher or lower potassium levels.⁹ Patel RB et al evaluated the association between potassium levels, cardiac arrhythmias, and cardiovascular death in patients with non-ST-segment elevation myocardial infarction or unstable angina. Potassium levels were measured in 6515 patients prior to randomization to receive either ranolazine or a placebo in the MERLIN-TIMI 36 trial. A seven-day continuous

electrocardiographic assessment was obtained to determine the incidence of non-sustained ventricular tachycardia (NSVT) and ventricular pauses. The association between potassium levels and cardiovascular death was evaluated using a Cox proportional hazards regression model with multivariable adjustment. NSVT lasting for at least eight consecutive beats occurred more frequently at potassium levels <3.5 mEq/L than at potassium levels \geq 5 mEq/L (10.1 vs. 4.5%, $p=0.03$ for trend), whereas the inverse pattern was observed for ventricular pauses >3 s, which occurred more frequently at potassium levels \geq 5 mEq/L than at potassium levels <3.5 mEq/L (5.9 vs. 2.0%, $p=0.03$ for trend). There was a U-shaped relationship between the potassium level at admission and both early and late risk of cardiovascular death. Compared with patients with potassium levels of 3.5 to <4 mEq/L, a potassium level <3.5 mEq/L was associated with an increased risk of cardiovascular death at day 14 (2.4 vs. 0.8%, HRadj 3.1, $p=0.02$) and at one year (6.4 vs. 3.0%, HRadj 2.2, $p=0.01$). The risk of cardiovascular death at one year was also significantly increased at potassium levels \geq 4.5 mEq/L and a similar trend was noted at potassium levels \geq 5 mEq/L. The lowest risk of cardiovascular death was observed in patients with admission potassium levels between 3.5 and 4.5 mEq/L.¹⁰ Colombo MG et al examined the association between SPC and long-term mortality following AMI in patients recruited from a population-based registry. examined the association between SPC and long-term mortality following AMI in patients recruited from a population-based registry. Patients were categorized into five SPC groups (<3.5, 3.5 to <4.0, 4.0 to <4.5, 4.5 to <5.0, and \geq 5.0 mEq/l). The outcome of the study was all-cause mortality. Cox regression models adjusted for risk factors, co-morbidities and in-hospital treatment were constructed. 249 patients (7.4%) had a low SPC (<3.5 mEq/l) and 134 (4.0%) patients had a high SPC (\geq 5.0 mEq/l). Patients with SPC of \geq 5.0 mEq/l had the highest long-term mortality (29.9%) and in the adjusted model, their risk of dying was significantly increased (HR 1.46, 95% CI 1.03 to 2.07) compared to patients with SPC between 4.0 and <4.5 mEq/l. Analyses of increasing observation periods showed a trend towards a higher risk of dying in patients with SPC between 4.5 and <5.0 mEq/l. An admission SPC of \geq 5.0 mEq/l might be associated with an increased mortality risk in patients with AMI.¹¹

CONCLUSION

Under the light of above obtained data, the authors conclude that electrolyte disturbance might contribute towards the pathophysiology of AMI. However; further studies are recommended.

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